

Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
L1	26	(traumatic) near3 (amnesia)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/12/08 16:50
L2	2	I1 and nootropic	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/12/08 16:48
L3	160	(traumatic or trauma or shock) near3 (amnesia)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/12/08 16:50
L4	36	I3 and nootropic	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/12/08 16:50
L5	1	I3 and nootropic and proline	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/12/08 16:51
L6	1	I4 and proline	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/12/08 16:52

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NEWS EXPRESS DECEMBER 02 CURRENT VERSION FOR WINDOWS IS V8.01,  
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FULL ESTIMATED COST	0.06	0.27

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=> file biosis embase medline caplus

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.21	0.48

FILE 'BIOSIS' ENTERED AT 15:27:34 ON 08 DEC 2005

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FILE 'MEDLINE' ENTERED AT 15:27:34 ON 08 DEC 2005

FILE 'CAPLUS' ENTERED AT 15:27:34 ON 08 DEC 2005

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=> nootropic (3a) trauma

L1 4 NOOTROPIC (3A) TRAUMA

=> dup rem l1

PROCESSING COMPLETED FOR L1

L2 4 DUP REM L1 (0 DUPLICATES REMOVED)

=> d ibib abs totatl

'TOTATL' IS NOT A VALID FORMAT

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individual files.

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L2 ANSWER 1 OF 4 CAPLUS COPYRIGHT 2005 ACS on STN

AN 2003:168111 CAPLUS

DN 139:286165

TI Neuroprotective and Nootropic Actions of a Novel Cyclized Dipeptide After  
Controlled Cortical Impact Injury in Mice

AU Faden, Alan I.; Fox, Gerard B.; Di, Xiao; Knoblach, Susan M.; Cernak,  
Ibolja; Mullins, Paul; Nikolaeva, Maria; Kozikowski, Alan P.

CS Dep. Neurosci., Georgetown Univ. Med. Cent., Washington, DC, 20057, USA

SO Journal of Cerebral Blood Flow and Metabolism (2003), 23(3), 355-363

CODEN: JCBMDN; ISSN: 0271-678X

PB Lippincott Williams & Wilkins

DT Journal  
LA English  
RE.CNT 34 THERE ARE 34 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d ibib abs total

L2 ANSWER 1 OF 4 CAPLUS COPYRIGHT 2005 ACS on STN  
ACCESSION NUMBER: 2003:168111 CAPLUS  
DOCUMENT NUMBER: 139:286165  
TITLE: Neuroprotective and Nootropic Actions of a Novel  
Cyclized Dipeptide After Controlled Cortical Impact  
Injury in Mice  
AUTHOR(S): Faden, Alan I.; Fox, Gerard B.; Di, Xiao; Knoblach,  
Susan M.; Cernak, Ibolja; Mullins, Paul; Nikolaeva,  
Maria; Kozikowski, Alan P.  
CORPORATE SOURCE: Dep. Neurosci., Georgetown Univ. Med. Cent.,  
Washington, DC, 20057, USA  
SOURCE: Journal of Cerebral Blood Flow and Metabolism (2003),  
23(3), 355-363  
CODEN: JCBMDN; ISSN: 0271-678X  
PUBLISHER: Lippincott Williams & Wilkins  
DOCUMENT TYPE: Journal  
LANGUAGE: English  
AB 1-ARA-35b (35b) is a cyclized dipeptide that shows considerable  
neuroprotective activity and improves neurol. recovery after fluid  
percussion-induced traumatic brain injury in rats. The authors evaluated  
the effects of treatment with 35b in mice subjected to controlled cortical  
impact brain injury. Animals treated with i.v. 35b after traumatic injury  
showed significantly enhanced recovery of beam walking and place learning  
functions compared with vehicle-treated controls, in addition to reduced  
lesion vols. Beneficial effects were dose related and showed an inverted  
U-shaped dose-response curve between 0.1 and 10 mg/kg. Protective actions  
were found when the drug was administered initially at 30 min or 1, 4, or  
8 h, but not at 24 h, after trauma. In sep. expts., rats treated with 35b  
on days 7 through 10 after injury showed remarkably improved place  
learning in comparison with injured controls. These studies confirm and  
extend the neuroprotective effects of this diketopiperazine in traumatic  
brain injury. In addition, they show that 35b has a relatively wide  
therapeutic window and improves cognitive function after both acute and  
chronic injury.  
REFERENCE COUNT: 34 THERE ARE 34 CITED REFERENCES AVAILABLE FOR THIS  
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 2 OF 4 CAPLUS COPYRIGHT 2005 ACS on STN  
ACCESSION NUMBER: 1998:468288 CAPLUS  
DOCUMENT NUMBER: 129:170390  
TITLE: The effect of nootropics on the function of brain  
mitochondria during the course of craniocerebral  
trauma in immature rats  
AUTHOR(S): Novikov, V. E.; Kovaleva, L. A.  
CORPORATE SOURCE: Smolensk. Gos. Med. Akad., Smolensk, 214019, Russia  
SOURCE: Eksperimental'naya i Klinicheskaya Farmakologiya  
(1998), 61(2), 65-68  
CODEN: EKFAE9; ISSN: 0869-2092  
PUBLISHER: Izdatel'stvo Folium  
DOCUMENT TYPE: Journal  
LANGUAGE: Russian  
AB A craniocerebral trauma was modeled in expts. on one-month-old rats.  
Oxidative phosphorylation in the brain mitochondria was studied by  
polarog. 1, 4, 7 days and 4 wk after the trauma. In the posttraumatic  
period the animals received piracetam (1 g/kg), picamilon (500 mg/kg),  
pyriditol (100 mg/kg), pantogam (160 mg/kg), ACTG (5 -10) (0.7 mg/kg),  
nooglutyl (25 mg/kg), and GVS (0.5 mg/kg). It was found that piracetam,

picamilon, and nooglutyl have a protective effect on the function of the brain mitochondria during the course of a craniocerebral trauma. Nooglutyl surpasses all the other drugs in its effect on the oxidative phosphorylation in mitochondria in immature rats during the posttraumatic period.

L2 ANSWER 3 OF 4 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 1997:529186 CAPLUS  
DOCUMENT NUMBER: 127:229508  
TITLE: The effect of agents with nootropic activity on oxidative phosphorylation in brain mitochondria in acute craniocerebral trauma  
AUTHOR(S): Novikov, V. E.; Kovaleva, L. A.  
CORPORATE SOURCE: Smolensk State Medical Academy, Smolensk, 214019, Russia  
SOURCE: Eksperimental'naya i Klinicheskaya Farmakologiya (1997), 60(1), 59-61  
CODEN: EKFAE9; ISSN: 0869-2092  
PUBLISHER: Izdatel'stvo Folium  
DOCUMENT TYPE: Journal  
LANGUAGE: Russian

AB An open craniocerebral trauma was simulated in rat expts. Oxidative phosphorylation in the brain mitochondria was studied by polygraphy 24 h after the trauma. It was found that trauma to the brain leads to inhibition of respiration in mitochondria in various metabolic states. Nooglutil in a dose of 50 mg/kg prevents these changes. Nooglutil is more effective than picamilon (500 mg/kg) and piriditol (100 mg/kg).

L2 ANSWER 4 OF 4 MEDLINE on STN

ACCESSION NUMBER: 90022023 MEDLINE  
DOCUMENT NUMBER: PubMed ID: 2572097  
TITLE: [Effect of nootropic agents on the brain bioelectrical activity and on the indices of neuromediator metabolism in the acute period of severe craniocerebral trauma]. Vliianie nootropov na bioelektricheskuu aktivnost' mozga i pokazateli neiromediatornogo obmena v ostrom periode tiazheloi cherepno-mozgovoï travmy.  
AUTHOR: Madorskii S V; Potapov A A; Piasetskaia M V; Shapova E V; Il'icheva R F  
SOURCE: Zhurnal voprosy neirokhirurgii imeni N. N. Burdenko, (1989 May-Jun) (3) 29-35.  
Journal code: 7809757. ISSN: 0042-8817.  
PUB. COUNTRY: USSR  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: Russian  
FILE SEGMENT: Priority Journals  
ENTRY MONTH: 198911  
ENTRY DATE: Entered STN: 19900328  
Last Updated on STN: 20000303  
Entered Medline: 19891109

AB Clinico-biochemical examination and EEG were conducted in 39 patients with severe craniocerebral **trauma** who were given **nootropic** agents in a complex of intensive therapy measures. Four types of changes of monoamine metabolism in treatment with piracetam were revealed which were combined with two types of EEG changes. The authors recommend the time for beginning piracetam therapy depending on the level of traumatic injury to the brain.

=>

=> amnesia (3a) (trauma or traumatic)

L3 784 AMNESIA (3A) (TRAUMA OR TRAUMATIC)

=> nootropic and 13

L4 0 NOOTROPIC AND L3

=> peptide and l3

L5 0 PEPTIDE AND L3

=> neuroprotective and l3

L6 0 NEUROPROTECTIVE AND L3

=> l3 and treatment

L7 106 L3 AND TREATMENT

=> dup rem l7

PROCESSING COMPLETED FOR L7

L8 68 DUP REM L7 (38 DUPLICATES REMOVED)

=> d

L8 ANSWER 1 OF 68 CAPLUS COPYRIGHT 2005 ACS on STN

AN 2005:1001826 CAPLUS

DN 143:279428

TI Methods for activating muscle-specific gene dmK protein kinase for  
enhancing learning and memory in recovery from stroke and  
**treatment** of neurological disorders

IN Kleim, Jeffrey; Alberini, Cristina; Garcia-Osta, Ana

PA Mount Sinai School of Medicine of New York University, USA

SO PCT Int. Appl., 75 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2005084184	A2	20050915	WO 2004-US5006	20040220
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,				
	CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,				
	GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC,				
	LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI,				
	NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY,				
	TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ,				
	BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE,				
	ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK,				
	TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
PRAI	WO 2004-US5006		20040220		

=> l8 and nootropic

L9 0 L8 AND NOOTROPIC

=> l8 and tripeptide

L10 0 L8 AND TRIPEPTIDE

=> l8 and proline

L11 0 L8 AND PROLINE

=> l8 and "amino acid"

<-----User Break----->

SEARCH ENDED BY USER

L12 3 L8 AND "AMINO ACID"

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(FILE 'HOME' ENTERED AT 15:27:07 ON 08 DEC 2005)

FILE 'STNGUIDE' ENTERED AT 15:27:13 ON 08 DEC 2005

FILE 'HOME' ENTERED AT 15:27:18 ON 08 DEC 2005

FILE 'BIOSIS, EMBASE, MEDLINE, CAPLUS' ENTERED AT 15:27:34 ON 08 DEC 2005

L1 4 NOOTROPIC (3A) TRAUMA  
L2 4 DUP REM L1 (0 DUPLICATES REMOVED)  
L3 784 AMNESIA (3A) (TRAUMA OR TRAUMATIC)  
L4 0 NOOTROPIC AND L3  
L5 0 PEPTIDE AND L3  
L6 0 NEUROPROTECTIVE AND L3  
L7 106 L3 AND TREATMENT  
L8 68 DUP REM L7 (38 DUPLICATES REMOVED)  
L9 0 L8 AND NOOTROPIC  
L10 0 L8 AND TRIPEPTIDE  
L11 0 L8 AND PROLINE  
L12 3 L8 AND "AMINO ACID"

=> l8 and review

L13 10 L8 AND REVIEW

=> d ibib abs total

L13 ANSWER 1 OF 10 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN  
ACCESSION NUMBER: 2001:136773 BIOSIS  
DOCUMENT NUMBER: PREV200100136773  
TITLE: Methotrimeprazine in the **treatment** of agitation  
in acquired brain injury patients.  
AUTHOR(S): Maryniak, Oleh [Reprint author]; Manchanda, Rahul; Velani,  
Asif  
CORPORATE SOURCE: Parkwood Hospital, 801 Commissioners Road East, London, ON,  
N6C 5J1, Canada  
olemar.medart@sympatico.ca  
SOURCE: Brain Injury, (February, 2001) Vol. 15, No. 2, pp. 167-174.  
print.  
ISSN: 0269-9052.  
DOCUMENT TYPE: Article  
LANGUAGE: English  
ENTRY DATE: Entered STN: 14 Mar 2001  
Last Updated on STN: 15 Feb 2002

AB Medical management of the agitation associated with acquired brain injury (ABI) has been problematic. At least 12 distinct drugs are currently recommended in the medical literature. In recent years, on the ABI in-patient rehabilitation unit, methotrimeprazine (MTZ) has come to be the preferred drug and is used routinely for effective **treatment** of agitation. The objective of this paper is to describe the use and safety of MTZ in the rehabilitation of ABI patients. A retrospective chart **review** of all patients discharged from the ABI unit over a course of 2 years was conducted. In addition to demographics such a aetiology of ABI, sex, age, length of stay, Glasgow Coma Scale, length of post-**traumatic amnesia** and others, a detailed analysis was made of the multidisciplinary progress notes to determine the daily agitation status and the daily use of psychotropic medication. All notes on side effects and adverse reactions were carefully documented. 120 first admission recent ABI patients were discharged in the 2-year study period. Of these, 69 (57%) had some level of agitation and 56 (48%) were treated with MTZ, in doses of 2-50 mg up to four times daily. Agitation was controlled in most cases. In only two cases were significant side effects noted. While MTZ has been used as a safe and effective neuroleptic in psychiatry for over 40 years, this is the first report of its use in treating agitation in ABI.

L13 ANSWER 2 OF 10 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN  
ACCESSION NUMBER: 1997:299197 BIOSIS  
DOCUMENT NUMBER: PREV199799598400

TITLE: Mild head injury: Neuropathology, sequelae, measurement and recovery.  
AUTHOR(S): King, Nigel  
CORPORATE SOURCE: Clinical Psychol. Dep., Rivermead Rehabilitation Cent., Abington Rd., Oxford OX1 4XD, UK  
SOURCE: British Journal of Clinical Psychology, (1997) Vol. 36, No. 2, pp. 161-184.  
ISSN: 0144-6657.  
DOCUMENT TYPE: Article  
General Review; (Literature Review)  
LANGUAGE: English  
ENTRY DATE: Entered STN: 9 Jul 1997  
Last Updated on STN: 9 Jul 1997

AB Head injuries are common in industrialized countries and the majority of them are defined as 'minor' or 'mild' injuries (MHI). These terms, however, can be misleading because the sequelae that often follow such injuries can cause significant detriment to psychosocial and interpersonal functioning. Clinical psychologists in most areas of specialism are likely to encounter MHI because of their high frequency and the types of problems they can cause. An overview of the body of knowledge on this subject is therefore of some importance. This paper **reviews** the literature concerning the neuropathology, measurement, sequelae and recovery of MHI. The following subjects are addressed: (i) the relationship between the neuropathology of severe head injury and the neuropathology of MHI; (ii) the limitations of traditional measures of head injury severity (e.g. post-traumatic amnesia) when applied to MHI; (iii) factors relevant to the recovery of post-concussion symptoms following MHI; and (iv) intervention and **treatment** following MHI.

L13 ANSWER 3 OF 10 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN  
ACCESSION NUMBER: 1992:77508 BIOSIS  
DOCUMENT NUMBER: PREV199293045963; BA93:45963  
TITLE: IS ROUTINE COMPUTED TOMOGRAPHY SCANNING TOO EXPENSIVE FOR MILD HEAD INJURY?.  
AUTHOR(S): STEIN S C [Reprint author]; O'MALLEY K F; ROSS S E  
CORPORATE SOURCE: SUITE 411, 3 COOPER PLAZA, CAMDEN, NJ 08103, USA  
SOURCE: Annals of Emergency Medicine, (1991) Vol. 20, No. 12, pp. 1286-1289.  
ISSN: 0196-0644.  
DOCUMENT TYPE: Article  
FILE SEGMENT: BA  
LANGUAGE: ENGLISH  
ENTRY DATE: Entered STN: 2 Feb 1992  
Last Updated on STN: 2 Feb 1992

AB Objective: To compare relative costs of treating mildly head-injured patients by routine admission or by using skull radiographs or cranial computed tomography (CT) scanning to screen patients for admission. Design: Retrospective record **review**, hypothetical costs based on actual patient course and requirements. Setting: Southern New Jersey Regional Trauma Center at Cooper Hospital/University Medical Center [Camden, New Jersey, USA]. Participants: 658 consecutive mildly head-injured patients admitted from 1986 to 1988. All were given cranial CT scans. Measurements: Records were reviewed retrospectively and hypothetical costs were calculated based on actual length of hospitalization, surgical intervention, etc. These costs were compared for different **treatment** protocols. Main results: The average cost if every patient had been admitted for observation given skull radiographs, with CT scans done on those exhibiting skull fracture or later deterioration, was \$1,207. If the CT scan had been used to identify patients with intracranial lesions and the others had been discharged, costs would have been almost 10% less. Had skull radiography been used to screen admissions, costs would have been 22% below those of routine CT scanning. However, these small savings are likely to be reduced by additional expenses related to missed intracranial lesions. Conclusions:



Every patient with loss of consciousness or post-**traumatic amnesia** should have routine CT scanning. If the scan is normal and there are no other reasons for admission, the patients can be discharged safely from the emergency department. This represents optimal care from a medical standpoint and is justified from a cost-effectiveness point of view.

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ACCESSION NUMBER: 2005273463 EMBASE  
TITLE: Overview of traumatic brain injury patients at a tertiary trauma centre.  
AUTHOR: De Guise E.; Feyz M.; Leblanc J.; Richard S.-L.; Lamoureux J.  
CORPORATE SOURCE: E. De Guise, Montreal General Hospital/MUHC, 1650 Avenue Cedar, Montreal, Que. H3G 1A4, Canada  
SOURCE: Canadian Journal of Neurological Sciences, (2005) Vol. 32, No. 2, pp. 186-193.  
Refs: 41  
ISSN: 0317-1671 CODEN: CJNSA2  
COUNTRY: Canada  
DOCUMENT TYPE: Journal; General Review  
FILE SEGMENT: 008 Neurology and Neurosurgery  
017 Public Health, Social Medicine and Epidemiology  
019 Rehabilitation and Physical Medicine  
LANGUAGE: English  
SUMMARY LANGUAGE: English; French  
ENTRY DATE: Entered STN: 20050707  
Last Updated on STN: 20050707

AB Objective: The goal of this study was to provide a general descriptive and cognitive portrait of a population with traumatic brain injury (TBI) at the time of their acute care stay. Material and methods: Three hundred and forty-eight TBI patients were assessed. The following data were collected for each patient: age, level of education, duration of post-**traumatic amnesia**, Galveston Orientation **Amnesia** Test score, Glasgow Coma Scale score, results of cerebral imaging, Neurobehavioral Rating Scale score, the Functional Independence Measure cognitive score and the Glasgow Outcome Scale score. Results: The clinical profile of the population revealed a mean age of 40.2 ( $\pm 18.7$ ) and a mean of 11.5 ( $\pm 3.6$ ) years of education. Most patients presented with frontal (57.6%) and temporal (40%) lesions. Sixty-two percent had post-**traumatic amnesia** of less than 24 hours. Seventy percent presented with mild TBI, 14% with moderate and 15% with severe TBI. The cognitive deficits most frequently observed on the Neurobehavioral Rating Scale were in the areas of attention, memory and mental flexibility as well as slowness and mental fatigability. Most patients had good cognitive outcome on the Functional Independence Measure and scores of 2 and 3 were frequent on the GOS. Forty-five percent of the patients returned home after discharge, 51.7% were referred to in or out patient rehabilitation and 3.2% were transferred to long-term care facilities. Conclusion: Because of the specialized mandate of acute care institutions, the information provided here concerning characteristics of our TBI population is essential for more efficient decision-making and planning/programming with regards to care and service delivery.

L13 ANSWER 5 OF 10 EMBASE COPYRIGHT (c) 2005 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2002185725 EMBASE  
TITLE: Combat-induced dissociative amnesia: **Review** and case example of generalized dissociative amnesia.  
AUTHOR: Witztum E.; Margalit H.; van der Hart O.  
CORPORATE SOURCE: Dr. E. Witztum, Mental Health Center, Ben-Gurion University of the Negev, Faculty of Health Sciences, P.O. Box 4600, Beer-Sheva 84170, Israel. elyit@actcom.co.il  
SOURCE: Journal of Trauma and Dissociation, (2002) Vol. 3, No. 2,

pp. 35-55.

Refs: 63

ISSN: 1529-9732 CODEN: JTDOAJ

COUNTRY: United States

DOCUMENT TYPE: Journal; General Review

FILE SEGMENT: 032 Psychiatry

035 Occupational Health and Industrial Medicine

LANGUAGE: English

SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 20020613

Last Updated on STN: 20020613

AB Dissociative **amnesia** following combat **trauma** in various wars has been extensively documented. In this article, we describe theoretical constructs related to dissociative amnesia, and integrate them with clinical practice through the presentation of a case. Although there is ample documentation of this condition in combat soldiers, in actual clinical practice such dissociative amnesia is probably underdiagnosed and undertreated. This may be detrimental to therapeutic progress, given the fact that ongoing memory deficits constitute one of the core symptoms of chronic PTSD in combat veterans. As illustrated in our case example of combat-induced generalized dissociative amnesia, combat-induced amnesia may also reflect previously existing dissociated traumatic memories that become reactivated during trauma. In this case, intensive **treatment** using hypnosis within a larger therapeutic milieu involved both the uncovering and processing of recent dissociated traumatic experiences, and, by necessity, other traumas of the past. .COPYRGT. 2002 by The Haworth Press, Inc. All rights reserved.

L13 ANSWER 6 OF 10 EMBASE COPYRIGHT (c) 2005 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2001064621 EMBASE

TITLE: To do or not to do? Magnetic resonance imaging in mild traumatic brain injury.

AUTHOR: Voller B.; Auff E.; Schnider P.; Aichner F.

CORPORATE SOURCE: F. Aichner, O. O. Landes-Nervenlinik, Neurologische Abteilung, Wagner-Jauregg-Weg 15, A-4021 Linz, Austria. franz.aichner@wj.lkh.ooe.gv.at

SOURCE: Brain Injury, (2001) Vol. 15, No. 2, pp. 107-115.

Refs: 42

ISSN: 0269-9052 CODEN: BRAIEO

COUNTRY: United Kingdom

DOCUMENT TYPE: Journal; General Review

FILE SEGMENT: 014 Radiology

008 Neurology and Neurosurgery

027 Biophysics, Bioengineering and Medical Instrumentation

LANGUAGE: English

SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 20010301

Last Updated on STN: 20010301

AB Clinical quantification of mild traumatic brain injury (MTBI) patients should be based on Glasgow coma scale (GCS) score, duration of loss of consciousness (LOC) and post-**traumatic amnesia** (PTA). In addition, a short practicable neuropsychological test might be useful in detecting minor memory and attentional deficits. MRI appears to be the most sensitive imaging method for assessing MTBI so far, but information regarding a visualized lesion is not usually utilized in the classification of MTBI. Magnetic resonance imaging (MRI) should, therefore, play a major role in any MTBI classification scheme. An appropriate MRI protocol has to be chosen using at least T(1) weighted, T(2) weighted, proton density and gradient-echo (GRE) sequence images, all in at least two planes, in order to detect and classify all lesions precisely. Owing to the fact that acute lesions may be missed, it is advisable to perform MRI in the first 2 weeks following trauma. Further

research is necessary to clarify the relationship between chronic symptoms after MTBI and MRI abnormalities. It may, thus, be possible to provide optimal strategies for emergency department management, to define a group of patients with a need for acute and rehabilitative intervention after MTBI, and to predict their outcome.

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ACCESSION NUMBER: 2000306386 EMBASE  
TITLE: Controversy about brain damage following cranio-cervical acceleration-deceleration trauma.  
AUTHOR: Radanov B.P.  
CORPORATE SOURCE: Prof. B.P. Radanov, Psychiatric Poliklinik, Universitaet Bern, Ch-3010, Bern, Switzerland. radanov@pupk.unibe.ch  
SOURCE: Journal of Musculoskeletal Pain, (2000) Vol. 8, No. 1-2, pp. 179-192.  
Refs: 76  
ISSN: 1058-2452 CODEN: JMPAEQ  
COUNTRY: United States  
DOCUMENT TYPE: Journal; General Review  
FILE SEGMENT: 008 Neurology and Neurosurgery  
033 Orthopedic Surgery  
LANGUAGE: English  
SUMMARY LANGUAGE: English  
ENTRY DATE: Entered STN: 20000914  
Last Updated on STN: 20000914

AB Objectives: To ascertain the levels of trauma from an acceleration-deceleration event which would expose a person to brain trauma, and to suggest clinical guidelines to the evaluation of injury outcomes which would indicate significant brain trauma was a potential sequelae. Methods: The brain-injury literature that related to clinical, psychological, biomechanical, and imaging studies was reviewed and compared for correlation to injury outcomes. Results: The severity of cranio-cervical tissue damage is clearly correlated to the degree of acceleration-deceleration trauma exposure. The size of a brain lesion is in a direct correlation to the duration and depth of unconsciousness, and the duration of post-traumatic amnesia. The acceleration-deceleration mechanism of injury would expose the prefrontal, frontal, or temporal cortex to diffuse axonal injury. These brain regions are crucial for complex attentional functioning. Indicators of trauma severity [loss of consciousness] are correlated to both clinical [cognitive impairment] and neuro-imaging findings [PET]. Presumed head-neck trauma that leads to an unconscious period of less than 10 minutes, or an amnesia period that spans less than four hours [a minor concussion] is not likely to cause any lasting brain damage or dysfunctional mental sequelae based on brain trauma. If these relationships cannot be established, factors other than brain damage [pain, adverse effect of medication, alcohol, psychological-personality problems, other agendas] should be considered the basis of complaints following cranio-cervical acceleration-deceleration trauma. Conclusions: Minor trauma exposure without a significant unconscious period, including amnesia, is very unlikely to have caused diffuse axonal injury, nor brain trauma.

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ACCESSION NUMBER: 87012333 EMBASE  
DOCUMENT NUMBER: 1987012333  
TITLE: Post-traumatic hypopituitarism. Six cases and a review of the literature.  
AUTHOR: Edwards O.M.; Clark J.D.A.  
CORPORATE SOURCE: Department of Medicine, Addenbrooke's Hospital, Cambridge CB2 2QQ, United Kingdom  
SOURCE: Medicine, (1986) Vol. 65, No. 5, pp. 281-290.  
CODEN: MEDIAV

COUNTRY: United States  
DOCUMENT TYPE: Journal  
FILE SEGMENT: 003 Endocrinology  
006 Internal Medicine  
049 Forensic Science Abstracts  
LANGUAGE: English  
ENTRY DATE: Entered STN: 911211  
Last Updated on STN: 911211

AB The typical patient with post-traumatic hypopituitarism is a young adult male presenting months to years after an automobile accident, following which he was unconscious for several days. He will probably have sustained a fracture of the base of the skull and on recovery is likely to have permanent visual or other neurological sequelae. Temporary or permanent diabetes insipidus may have occurred. The features of panhypopituitarism such as weight loss, fatigue, faintness, loss of libido, and impotence may have been ascribed to depression or the 'postconcussion syndrome' and often inappropriate **treatment** and rehabilitation advised. The striking feature on **review** of the literature is that the pathological consequences of head injury to the pituitary and hypothalamus have been well described, while only 47 cases of traumatic hypopituitarism have been reported. The most likely reason for this disparity is that head injury of sufficient severity to cause hypothalamic and pituitary damage commonly led to death. More patients now survive owing to the availability of intensive care; accordingly, most cases have been reported in the last 15 years. However, several patients are described in whom the initiating head injury was not associated with a skull fracture or followed by coma. We recommend that patients with major head injury (defined by post-**traumatic amnesia** greater than 24 hours), and in particular those with fractures of the base of the skull or diabetes insipidus should be closely monitored for symptoms and signs of endocrine dysfunction and appropriate dynamic pituitary-function tests performed.

L13 ANSWER 9 OF 10 MEDLINE on STN  
ACCESSION NUMBER: 2003040482 MEDLINE  
DOCUMENT NUMBER: PubMed ID: 12547983  
TITLE: Evaluation and **treatment** of psychosis after traumatic brain injury.  
AUTHOR: McAllister Thomas W; Ferrell Richard B  
CORPORATE SOURCE: Dartmouth Medical School, Department of Psychiatry, Dartmouth Hitchcock Medical Center, One Medical Center Drive, Lebanon, NH 03756, USA..  
Thomas.W.McAllister@Dartmouth.edu  
CONTRACT NUMBER: R01 NS40472-01 (NINDS)  
SOURCE: NeuroRehabilitation, (2002) 17 (4) 357-68. Ref: 122  
Journal code: 9113791. ISSN: 1053-8135.  
PUB. COUNTRY: Netherlands  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
General Review; (REVIEW)  
LANGUAGE: English  
FILE SEGMENT: Priority Journals  
ENTRY MONTH: 200304  
ENTRY DATE: Entered STN: 20030128  
Last Updated on STN: 20030403  
Entered Medline: 20030402

AB A **review** of research studies to date suggests that psychosis is a relatively rare, but serious, complication of traumatic brain injury (TBI). Psychotic syndromes occur more frequently in individuals who have had a TBI than in the general population. Onset of symptoms can be early or late. Psychosis can occur during the period of post-**traumatic amnesia**, in association with post-traumatic epilepsy, in association with TBI-related mood disorders, and as a chronic, schizophrenia-like syndrome. TBI can interact with genetic vulnerability to increase the risk of developing illnesses such as schizophrenia. Thorough diagnostic assessment is the foundation of rational and effective

pharmacotherapy for psychosis after TBI. Atypical antipsychotic drugs have emerged as first line drugs for **treatment** of psychotic disorders from all causes, including TBI. Anticonvulsant, antidepressant or other drugs may also be needed in some cases. Medication approaches must be adjusted for the particular characteristics and vulnerabilities of the patient with a TBI.

L13 ANSWER 10 OF 10 MEDLINE on STN  
 ACCESSION NUMBER: 95179089 MEDLINE  
 DOCUMENT NUMBER: PubMed ID: 7874091  
 TITLE: Prediction of employment status 2 years after traumatic brain injury.  
 AUTHOR: Ponsford J L; Olver J H; Curran C; Ng K  
 CORPORATE SOURCE: Bethesda Hospital, Melbourne, Australia.  
 SOURCE: Brain injury : [BI], (1995 Jan) 9 (1) 11-20.  
 Journal code: 8710358. ISSN: 0269-9052.  
 PUB. COUNTRY: ENGLAND: United Kingdom  
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
 LANGUAGE: English  
 FILE SEGMENT: Priority Journals  
 ENTRY MONTH: 199504  
 ENTRY DATE: Entered STN: 19950419  
 Last Updated on STN: 19950419  
 Entered Medline: 19950406

AB The present study used a multivariate approach to investigate which of a range of variables relating to demographic factors, injury severity and degree of disability on admission to rehabilitation were the best predictors of employment status 2 years after traumatic brain injury (TBI). Subjects were 74 TBI patients who had been working prior to injury, had undergone rehabilitation at Bethesda Hospital and attended a **review** clinic 2 years after injury. A cross-validation sample consisted of a further 50 such subjects. Following preliminary analysis four input variables were selected: age under or over 40 at time of injury, Glasgow Coma Scale score on acute hospital admission, duration of post-traumatic **amnesia** and total score on the Disability Rating Scale (DRS) on admission to rehabilitation. Stepwise discriminant function analysis resulted in a discriminant function consisting of three variables--total score on the Disability Rating Scale, Glasgow Coma Scale Score and age--which correctly classified 74% of grouped cases. A second analysis using the original discriminant function correctly classified 68% of the cross-validation sample. Chi-square analysis showed no significant difference between these results, thus confirming these variables, in combination, as predictors of employment status 2 years after TBI.

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